dance of $O_2(^1\Sigma_g)$ might be adequate, dayglow measurements of emission from this state (*2*) show that the actual abundance above 50 km is less than 10^{13} cm^{-2}. Even with fully allowed absorption, the abundance is much too small to yield the observed effect.

As Krueger points out, measurement of the $O_2(^1\Delta_g)$ dayglow at 1.27 μ (*3*) shows that the abundance of this excited state is compatible with a 10^{-17} cm^2 cross section. But if absorption by $O_2(^1\Delta_g)$ were as strong as this, then certain unacceptable consequences arise. The absorption appears between 2900 and 3100 Å and includes the threshold wavelength for dissociation, 3037 Å; it is thus energetically possible for the process to destroy $O_2(^1\Delta_g)$. Where dissociation does not occur, the process will lead to fluorescent scattering and a "dayglow" whose intensity may be estimated. The incident solar flux over a band 100 Å wide at 3000 Å is about 10^{15} photons cm^{-2} sec^{-1} (*4*), and an absorption coefficient of 10^{-17} cm^2 implies a scattering of 10^{-2} photon molecule^{-1} sec^{-1}. This probability of scattering is about 30 times larger than the probability of emission (at 1.27 μ) from $O_2(^1\Delta_g)$ (*5*). It follows from the dayglow abundance (*3*) that the total scattered intensity above 50 km would be about 600 megarayleighs; above 80 km the intensity would be still about 30 megarayleighs. As an extreme case we assume that the fluorescent scattering is uniformly distributed over the visible region; the sky brightness at 50 km would then be about 200 kr/Å and at 80 km it would be 10 kr/Å. In both cases the predicted intensity is roughly 100 times larger than that expected for Rayleigh scattering above the same altitude. Rocket measurements (*6*) at several wavelengths between 4000 and 7000 Å make it clear that above 70 km the sky intensity is in fact close to the Rayleigh value. If the scattering were confined to the same wavelength region as the absorption, the sky intensity at 3000 Å would be as strong at 50 km as it is at ground level. The intensity at 80 km would be orders of magnitude larger than that measured by Barth (*7*) in this wavelength region.

Scattering would have to be important for all wavelengths in the absorption band which do not lead to dissociation. If the scattering process reforms $O_2(^1\Delta_g)$ it is reasonable to expect scattered wavelengths somewhere near 3000 Å or, possibly, at somewhat longer wavelength in the visible or near infrared. Since the scattered intensity appears to be unacceptably high we may consider the alternative process which destroys $O_2(^1\Delta_g)$. The foregoing computation shows that, if destruction were the sole outcome of the absorption process, the effective lifetime of $O_2(^1\Delta_g)$ would be 30 times smaller than the radiative lifetime. A discussion of the daytime $O_2(^1\Delta_g)$ measurements (*3*) indicates that no production mechanism yet conceived is capable of accounting for the measured abundance of $O_2(^1\Delta_g)$ above 70 km if the state is destroyed more rapidly than the rate set by spontaneous emission at 1.27 μ. It is therefore difficult to avoid the conclusion that the species responsible for the absorption at 3000 Å cannot be $O_2(^1\Delta_g)$; neither fluorescent scattering by the state nor significant destruction of it can be tolerated.

Rather similar arguments may be advanced against Krueger's third candidate. The argument against fluorescent scattering may be carried over if, as Krueger suggests, the absorption is in the Schumann-Runge band system, from O_2 molecules near $v = 11$ in the ground state. There is also a major problem in accounting for the required abundance of such highly vibrating O_2. Such molecules will doubtless be destroyed by collision-induced relaxation at a much faster rate than is $O_2(^1\Delta_g)$ which can withstand about 10^9 collisions with "air" molecules (*8*). It would therefore be necessary that a process exist which can create vibrationally excited O_2 at a rate that is orders of magnitude greater than the rate for $O_2(^1\Delta_g)$. The energy problems associated with such a requirement appear insurmountable when one considers that the creation of $O_2(^1\Delta_g)$ itself requires efficient use of the entire solar spectrum between 2000 and 3000 Å.

The measurements reported by Krueger resemble some described by Tohmatsu (*9*) in which rocket photometer results at 3000 Å implied a larger ozone abundance above 50 km than those from a photometer at 2500 Å; this appears to weaken the possibility of an instrumental error since the photometric methods were quite different. In both sets of measurements, the solar zenith angle was small and the optical depth of the excess absorption near 3000 Å was less than 1. Unless the absorber disappears before sunset one would expect that measurements of the solar spectrum at large zenith angle should reveal almost complete extinction in the vicinity of 3000 Å even up to 60 km. There appears to be no such anomalous absorption present in the early solar spectra taken near sunset at such heights (*10*) many years ago. It would be of interest to have additional measurements of the phenomenon at large solar zenith angle.

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Bladder Tumorigenesis

Bladder tumors have been induced in rats by a number of industrial chemicals and by cyclamate. There is, however, one factor implicated in bladder tumorigenesis in rats which is frequently ignored. We refer to the possible parasitism among the rats used in these studies by the bladder nematode *Trichosomoides crassicauda* and to the possible potentiative effect of this parasite on the chemical induction of bladder tumors.

The adult *T. crassicauda* resides in the bladder, renal pelvis, and ureter, usually stimulating little tissue reaction. Parasitism with this nematode is endemic in wild rats (*1*). In 1964 Chapman (*2*) examined rats from 19 commercial American sources and found that approximately one-half of the groups contained at least one parasitized animal. The incidence of infection in these groups varied from 4 to 91 percent (11 to 24 rats per group). Although a few cesarian-delivered rats were free of parasites, one cannot dismiss the possibility of transplacental infection or subsequent postnatal infection. Dissemination of infection occurs readily since embryo-

nated eggs that are passed in the urine are immediately infective. Contaminated cages, unless washed thoroughly with sufficiently hot water, can cause a large proportion of a colony to become infected.

A report by Chapman (*3*) indicates that infection with *T. crassicauda* may increase the incidence of bladder tumors in rats fed the well-known bladder carcinogen 2-acetylaminofluorene. A somewhat dated controversy as to whether *T. crassicauda* is associated, in the absence of exogenous carcinogens, with bladder tumors (*3*, p. 154) need not be invoked.

Thus it would appear desirable that any investigator encountering bladder tumors in rats make a thorough search for this parasite. Methods are available for eliminating the infection and for maintaining a clean rat colony (*4*).

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Reticular Stimulation and Chlorpromazine

Based on the hypothesis that schizophrenics are overaroused as a result of long-term activation of the brainstem reticular formation (*1*), Kornetsky and Eliasson proposed that animals electrically stimulated in the reticular formation are overaroused in a similar fashion (*2*). They postulated an "inverted U" model in which overarousal moves subjects beyond an optimum level of performance and chlorpromazine keeps subjects before the optimum point; therefore the drug reduces the overarousal effects and produces improved performance. They tested their hypothesis on three rats in a test of sustained attention and found fewer errors in performance when intermittent stimulation and chlorpromazine were combined than with either drug or stimulation alone. We have made slightly different tests with rats given chlorpromazine and electrical reticular stimulation and have found similar results at certain doses, but the effect was more marked with a barbiturate combined with stimulation which suggests that the "inverted U" hypothesis is not specific to chlorpromazine.

We used 82 adult male Wistar rats with silver wire electrodes (0.015 cm in diameter) permanently implanted (*3*) in the mesencephalic reticular formation according to the stereotaxic coordinates of de Groot (*4*); experiments were begun at least 1 week after surgery. Square-wave pulses were applied at currents ranging from 25 to 80 μa and at a frequency of 300 hz of 1-msec duration (*5*). At the end of each experiment the animals were killed and the brains perfused for histological examination. The sites of stimulation were in the dorsal lateral area of the mesencephalic reticular formation [A 0.6 to A 2.2 (*4*)].

The first experiment was a maze-running test in which rats deprived of water for 23 hours were taught to run to the end of an arm (35.5 cm long) of a Y-maze to obtain 0.5 ml of water. To get another reward the rats had to run to the end of the next arm of the maze on the right. A manual correction procedure was used in training. In this way rats learned to run a clockwise route around the maze. Errors were scored when rats took the wrong alley, and the total number of entries made gave a measure of general activity. The rats were trained daily, and, when they reached an asymptote level on three consecutive days with the same number of entries ± 2, the test conditions were applied for the following 3 days. Reticular stimulation was given throughout a 5-minute trial session, and drugs were injected 30 minutes before testing. Chlorpromazine was given in doses of 1, 2, and 4 mg/kg, and amylobarbitone in doses of 10, 20, and 40 mg/kg, all subcutaneously. There were 18 saline control animals, 9 of which were stimulated, and 10 rats in each drug dose group, half of which received stimulation. Four rats were eliminated because the electrodes were in the wrong site or because they did not reach the learning criterion. Reticular stimulation significantly reduced activity [$P < .01$ (*6*)] but had no significant effect on the number of errors made. Chlorpromazine produced a decline in activity, but in combination with stimulation there was no significant difference from the nonstimulated condition at a dose of 1 and 2 mg/kg. If we consider the detrimental effect with stimulation alone in the saline group, this result shows a reduction of the stimulation effect. At 4 mg/kg, however, there was a greater decrease in activity when stimulation was given than with the drug alone ($P < .025$). This potentiation of the drug and stimulation is anomalous with the proposed "inverted U" model (*2*). The medium dose plus stimulation was also anomalous in that it produced a significant interference with the accuracy of performance ($P < .025$). The barbiturate alone lowered activity, but in combination with stimulation there was a significant increase in activity above the level produced by the drug alone at all three doses (test of orthogonal contrasts, $t = 2.42$, 1,21 d.f., $P < .05$). The effect seen with amylobarbitone alone on the error score was also offset by the combination of barbiturate and stimulation at a dose of 40 mg/kg ($P < .025$). Thus there was an antagonism between both drugs and stimulation, but the effect produced by the amylobarbitone was greater than that of chlorpromazine.

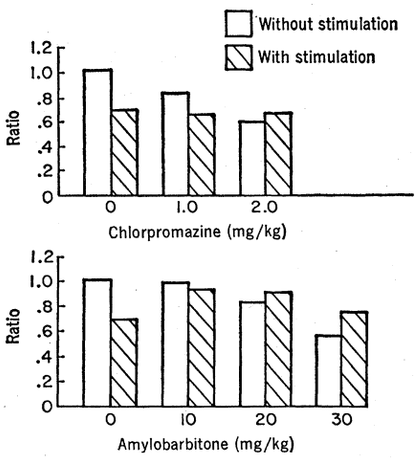

Fig. 1. The effect of drugs and reticular stimulation on discriminated conditioned avoidance responding expressed as a ratio (mean) of the test performance to the criterion performance level.

The next experimental test was set up to require little response activity but to be more a test of accuracy. A discriminated, operant-conditioned avoidance response to a flashing light was established in four rats. At the beginning of a trial the conditioned stimulus (CS) was presented. If a subject pressed the lever within 10 seconds, shock was avoided; if the lever was